

## Periscope.

### PATHOLOGY (INCLUDING PATHOLOGICAL ANATOMY) OF NERVOUS SYSTEM.

**Recent Observations on Degeneration, and on Nerve Tracts in the Spinal Cord.—A Critical Account.** By J. N. LANGLEY, M.A., F.R.S. *Brain*, April, 1886.

In view of the great importance of the facts collected and the conclusions reached by Langley, we propose to devote several pages to the consideration of his paper. It is a "critical digest" and does not well bear further condensation. For this reason we shall reprint the original (contrary to our usual custom) with but a few unimportant omissions. Langley's results are based upon articles by: 1, Langley and Sherrington; 2, Pitres; 3, Löwenthal; 4, Sherrington; 5, Homén.

The following may be taken as a rough summary of the chief points connected with the observation of secondary degeneration, although no doubt several of the statements are open to discussion. Secondary degeneration is best observed with the microscope four to five weeks after the injury. The degeneration can be made out near the lesion in a week or less. It spreads fairly rapidly—probably in less than twenty-four hours—along the whole course of the nerve fibres. It can be earliest seen by the alteration in the neural axis, a day or two later by the change in the myelin sheath also. In one to two weeks it can be observed by the eye in the cord hardened in potassium bichromate. In about three weeks it can be seen by the eye in the fresh cord. The neuroglia now begins to increase, and the nerve fibres to be absorbed, although probably none disappear for some weeks. In consequence of these changes, the degeneration is, from the sixth week onwards, more and more noted under the microscope by the change in the neuroglia, and less and less by the presence of altered nerve-fibres. In the lateral pyramidal tract altered nerve fibres are seen for three or four months. The length of time after the lesion during which the area of degeneration can be detected by the "scar," varies with the number of fibres which have degenerated; in the lateral pyramidal tract the scar can be seen for a

year, and probably for much longer. The degeneration can be seen in the fresh cord for four to five months ; during the last six weeks or so of this time, it changes from milky-white to grayish. Possibly in the cerebellar tract the change takes place earlier. In time (?six months) no change can be seen in the fresh cord. In the cord hardened in potassium bichromate, the area of degeneration becomes more distinct for four to six months ; later than this it becomes less and less distinct ; in the lateral pyramidal tract it may be seen for about a year. In its earlier stage, the area of degeneration may appear larger than it really is, in consequence of a spreading out of the connective tissue change ; in its later stage it becomes smaller, in consequence of a retraction (? partial disappearance) of the connective tissue.

Whilst all observers have paid considerable attention to the time after the lesion at which secondary degeneration makes its appearance, comparatively little attention has been paid to the time at which the degeneration of the nerve-fibres is complete. But this is a point of some importance. According to Löwenthal, the degenerating areas in the region of the lateral pyramidal tract, which result from sections of the cord in the cervical region, consist chiefly of altered fibres three months after the section. He finds, too, that the number of altered fibres is greater in twelve weeks than in six, and greater in six weeks than in three. Sherrington describes a few altered fibres as being present in the lateral pyramidal tract seven months after cortical lesion. But from the description of most observers, we should gather that some fibres disappear altogether in about a month. Homén, in three weeks, finds spaces from which fibres have disappeared ; but it is not clear whether the fibres had been absorbed from these spaces, or simply dropped out of the section. If fibres do disappear so early, and if Löwenthal's statement regarding the increase in the number of altered fibres for two to three months after the lesion is correct, it follows that some fibres have disappeared, whilst others have scarcely begun to degenerate. This seems extremely unlikely, if all the fibres are undergoing true secondary degeneration.

It seemed possible that there might be some differences between descending and ascending degeneration in these respects ; but I cannot find that any one who deals with both degenerations points out any difference. The only clear statement as to the time of disappearance of nerve-fibres in ascending degeneration which I have been able to find is given by Singer. According to him, in *young* dogs after section of the cord in the dorsal region, the nerve-fibres of the cerebellar tract and of the medial part of the posterior column completely disappear in four to five weeks. In the adult dog, the process is slower ; unfortunately he does not say how much slower. There seems to me, however, to be *primâ facie* ground for thinking that, at any rate in the pyramidal tract, some fibres do not begin to degenerate until several weeks after section of the cord.

Turning now to the nerve-tracts in the cord, we may first consider :

*The Pyramidal Tracts.*—Flechsig mentions that in the lateral pyramidal tracts there are a certain number of fibres which do not come from the opposite pyramid. But in addition to these fibres, it is probable that the lateral pyramidal tract, as described by Flechsig, contains many other fibres which do not belong to this tract.

Bouchard, in 1866, noticed that in man, the descending degeneration in the lateral column was considerably greater after injury to the medulla than after injury higher up in the brain.

Bouchard describes the area of degeneration which appears in the cord in the posterior part of the lateral column after injury to the medulla, as extending to the surface. Since the cerebellar tract occupies the periphery of this part of the cord, and does not undergo descending degeneration, Bouchard's statement regarding the peripheral strip probably rests on a misinterpretation of the appearance. According to Schiefferdecker, the sclerosis may extend beyond the area of the degenerated fibres ; to this the sclerosis seen by Bouchard at the periphery of the cord was probably due.

The fibres which degenerated after injury to the medulla, but not after injury to the brain, were situated on the lateral border of the pyramidal tract. These fibres he called "*fibres commissurales antérieures longues.*" This observation has passed with little or no notice.

A somewhat similar difference, however, is described by Löwenthal, as occurring in the dog, after section of the cord in the cervical region and after removal of the motor area of the cortex. According to Löwenthal, the long commissural fibres lie between the cerebellar tract and the lateral pyramidal tract ; they are chiefly large fibres ; whereas, as we know, the pyramidal tract fibres, like those of the pyramid, are chiefly moderate-sized and small. Löwenthal points out also that, after section of the cord in the cervical region, the degenerated area in the lateral column has a fairly sharp boundary on its lateral edge. In degeneration after removal of the cortex of the brain, this is certainly not the case, the degeneration becomes quite diffuse as it approaches the cerebellar tract.

Sherrington also has come to the conclusion that, at any rate in the cervical region of the cord, the real pyramidal tract occupies a part only of the pyramidal tract as figured by Flechsig. This conclusion is, however, based upon a comparison of the area of degeneration which he obtains in the dog, with the area of non-medullated fibres seen in the developing cord of the ape, and with the area of the pyramidal tract as given by Flechsig in the cord of man. The basis is somewhat uncertain. Proceeding, however, in this way, he concludes that the ventral and the latero-dorsal parts of Flechsig's pyramidal tract do not degenerate from cortical lesions, and hence are not continuations of the pyramid fibres.

On the whole, it seems probable that the pyramidal tract is much smaller than that given by Flechsig, that it does not form a compact bundle of fibres, but on its anterior (ventral), lateral, and posterior (dorsal) borders it becomes diffuse, its fibres being mixed with other fibres, many of which, especially on its lateral border, degenerate on section of the spinal cord. In its inner (mesial) side its fibres, as described by Flechsig become mixed with the small fibres of the "boundary layer." It will be noticed that, as far as Löwenthal's experiments extend, the "anterior long commissural fibres" might be "commissural" fibres of the cord. From Bouchard, however, we should conclude that they run from the medulla to the various segments of the cord, and hence we might call it the descending medullary tract.

*Bilateral Degeneration of Lateral Pyramidal Tract in Consequence of Unilateral Cortical Injury.*—It has been known for some time, that in man an injury confined to one side of the cord, might cause a degeneration in the lateral pyramidal tract of the opposite as well as in that of the same side of the cord. Charcot suggested as an explanation of this, that part of the lateral pyramidal tract crossed in the anterior commissure, especially in the upper dorsal region, to the lateral tract of the opposite side. Thus the fibres would first cross in the pyramidal decussation, and then again in the anterior commissure.

A case of bilateral degeneration in the cord of the dog, as the result of unilateral cortical injury, was described in 1880 by François-Franck and Pitres.

Moeli, in 1883, found after unilateral injury to the mid-brain in dogs, that besides the degeneration of the pyramidal tract of the opposite side there was a degeneration of a few scattered nerve fibres in the pyramidal tract of the same side.

An instance in the ape has also been described by Schäfer (1883); the examination was made seven months after the cortical injury. In the cervical region of the cord there was sclerosis in both lateral pyramidal tracts, the area of the two sides being about equal; but the sclerosis was less in intensity on the same side as the lesion, and on this side it could not be traced beyond the cervical region.

The bilateral degeneration of the pyramidal tract has received a good deal of attention from recent observers. In no case apparently does unilateral brain-injury cause degeneration of the opposite pyramid, so that whilst one pyramid only degenerates, there is degeneration in the region of both the lateral pyramidal tracts in the cord. We will, for lack of a better term, call the degeneration which occurs on the side of the cord opposite to the sound pyramid the *re-crossed degeneration*.

Pitres examined the spinal cord in forty cases of unilateral brain-injury in man. In ten of these there was on both sides sclerosis of the lateral pyramidal tracts. In some cases this could only be made out on microscopical examination. In six out of the ten cases the sclerosis was symmetrical and equal on the two

sides, and on both sides it occupied a larger area, chiefly in the ventral direction, than that occupied when sclerosis occurred on one side only. In the remaining four cases the re-crossed degeneration was of less intensity than that of the opposite side. According to Pitres, the re-crossed degeneration stretches throughout the cord, reaching the surface of the cord in the lumbar region. No relation could be made out between the presence or absence of a re-crossed degeneration, and the presence or absence of a degeneration in the medial part of the anterior column (the direct pyramidal tract, Türk's column). In six cases the direct pyramidal tracts showed some sclerosis; this, whilst varying in amount, was in all cases slight, and in one case only could it be traced in the lower dorsal and in the lumbar region. In three cases there was sclerosis in *both* direct pyramidal tracts.

Pitres considers the re-crossed degeneration to be due to the degeneration of fibres coming from the pyramid of the same side; *i. e.*, he extends to these fibres the view advanced by Bouchard and by Flechsig with regard to the direct pyramidal tract.

There are on this view four ways by which the fibres of each pyramid can proceed to their ending in the cord. They proceed along (1) the lateral pyramidal tracts of the opposite side; (2) the lateral pyramidal tract of the same side; (3) the direct pyramidal tract of the same side; (4) the direct pyramidal tract of the opposite side. Most commonly all the fibres take the first course; not infrequently they run both in the first and second courses; less frequently some fibres proceed by the third, the greater number running either in the first or in the first and second course; rarely some fibres may in addition take the fourth course. If this description is the true one, the term "re-crossed" degeneration, which I have used above, is obviously inapplicable; but there are reasons which will be mentioned later, against accepting Pitres' views. Charcot's suggestion, that the fibres of one lateral tract cross to the other by the anterior commissure, has, as Pitres points out, no satisfactory basis, for no degenerated fibres have as yet been described as occurring in the anterior commissure.

Bilateral degeneration in dogs after unilateral cortical injury has been described both by Löwenthal and by Sherrington.

Löwenthal mentions it as occurring in two out of about twenty cases. In each of the two the degeneration (sclerosis) was diffuse, and could not be traced farther than the mid-dorsal region. Sherrington found bilateral degeneration in each of twelve cases observed three to twelve months after the lesion, but found unilateral degeneration only, in two cases observed seven and fourteen days respectively after the lesion. The re-crossed degeneration appeared to him to be always somewhat less advanced than the degeneration on the opposite side of the cord. Hence he concludes that the re-crossed degeneration begins three or more weeks after the degeneration in the opposite lateral pyramidal tract. The conclusion is somewhat hasty; for in the two

cases in which re-crossed degeneration was absent, it might not have occurred had the animal been kept alive; and further, Sherrington does not appear to be quite certain that the nerve-fibres in the re-crossed degeneration were really in an earlier stage of degeneration than those on the opposite side of the cord. It may be noted, however, that if his conclusion is right, it disposes at once of the views both of Charcot and of Pitres as to the origin of the fibres in the re-crossed degeneration; for if the fibres which are affected in the re-crossed degeneration were directly continuous with the pyramid fibres, they would certainly not degenerate weeks later than the rest of the pyramid fibres in the lateral pyramidal tract.

Sherrington also finds that the re-crossed degeneration does not usually diminish regularly in its course down the cord, but is greater in the upper lumbar and lower dorsal region than in the mid-dorsal region, and is commonly greater between the third to seventh cervical nerve-roots—where, indeed, it is earliest seen—than between the first to third cervical nerve-roots. If this is the case the bilateral degeneration cannot be due to a crossed and an uncrossed portion of the pyramid, and the re-crossed degeneration cannot be due to pyramidal fibres. It is on this account that I have used the term “re-crossed degeneration.”

If the pyramids send no fibres to the lateral column of their own side, some other explanation of bilateral degeneration must be sought for. Let us consider in what other way it could be brought about. Since in the dog there is no evidence of a direct pyramidal tract, we have for the dog the simplest scheme, that all the pyramid fibres running to the cord decussate in the pyramidal decussation, and run down the cord in the lateral pyramidal tract of the opposite side. Since these fibres end in the gray substance of the cord, it is quite possible that their degeneration should cause more or less pronounced degeneration in some parts of the gray substance. And, in fact, one or two cases have been described of atrophy of cells in the anterior cornu in consequence of degeneration of the lateral pyramidal tract, and of atrophy of Clarke's column in consequence of degeneration of the posterior roots. And there is some evidence that in man the gray substance in lateral sclerosis is not infrequently affected in a less degree; for on the non-hemiplegic side it is not uncommon to find increase of reflexes, or general weakness without any discernible change in the columns of that side of the cord. These cases, although admitting of other explanation (see below), are most satisfactorily explained by supposing that the gray substance of the cord has been affected by the degeneration of the pyramid fibres. If the change proceeds further than this, viz., to degeneration, it is to be expected that some fibres proceeding from the gray substance to the columns of the cord, *i. e.*, connecting fibres, will degenerate. Such fibres, we may fairly conclude—bearing in mind the assumption with which we started—are the fibres of the re-crossed degeneration.

But if the disappearance of the pyramid fibres on one side can affect the gray substance sufficiently to lead to the degeneration of connecting fibres on the opposite side, we should certainly expect that it would cause some degeneration of connecting fibres on its own side. There seems to me to be some evidence that this does take place. We have seen above that there is some reason to believe that within the area of the lateral pyramidal tract some fibres show signs of degeneration very much earlier than others. This is not easy to explain, if all the fibres are the direct continuation of the fibres of the pyramid; whereas if the fibres which are first affected are pyramid fibres, and the later ones connecting fibres of the cord, the protracted period of degeneration presents no difficulty.

Sherrington and myself have used the term "tertiary" to denote that degeneration which is brought about by a change in gray substance as a consequence of nerve-fibres in it degenerating. We might then state the conclusions of the foregoing argument in the following way: injury to one lateral column of the cord (or unilateral injury to the motor area of the brain) causes secondary degeneration in the crossed pyramidal tract, and not infrequently tertiary degeneration of connecting fibres of the cord within the area of both crossed pyramidal tracts.

This view affords a basis for understanding how the re-crossed degeneration can, as described by Sherrington, be greater in the lower dorsal than in the mid-dorsal region; why it occurs later than the 'normal' degeneration; why on both sides of the cord fibres in an early stage of degeneration are found long after the injury; and why in bilateral degeneration the area is often, as described by Pitres, greater than in unilateral degeneration. It does not satisfactorily explain how the sclerosis can be—as described by Pitres in certain cases—equal in intensity on the two sides of the cord.

But with all this it must not be forgotten that the statements on which the view rests require confirmation; and, further, that so far no relation has been observed between bilateral degeneration and atrophy of gray substance. I have stated the view with some fulness, on account of its importance if true; but I need hardly say that I regard the questions here raised as at present quite open.

One other question of importance arises out of bilateral degeneration. This is whether each pyramid is connected with the gray substance of both sides of the spinal cord.

Flechsigs, in his "*Plan des menschlichen Gehirns*," figures the "direct" pyramidal tract as ending in the gray substance of the same side of the cord; those who accept Pitres' view of the meaning of bilateral degeneration might consider that here, too, each set of fibres ends in the gray substance of its own side.

That is to say, the extent of the uncrossed tracts of the pyramid would be a measure of the number of its fibres which end in

the cord on the same side as it. The variability of the course of the pyramid fibres in the cord would be associated with a variability in their termination.

It is clear that there is no necessity that this should be the case. All the fibres of one pyramid might run in one lateral column, and yet end in the gray substance of both sides of the cord; and, on the other hand, the fibres of one pyramid might run in both lateral and in both anterior columns, and yet end in the gray substance of one side only of the cord.

And that this is the case seems to me to be in the highest degree improbable. It requires a great deal of evidence even to make it likely that the individuals of one species vary so much, that in one the cortex of the brain is connected with one side only of the cord, and in another is connected with both sides; that, in fact, in different individuals the end stations of homologous fibres are indifferently on the right or on the left side of the body.

What, then, is the proof of this variation? It is partly that the pyramid fibres are said to vary in their course in the cord; this, even if we accept the fact, is, as I have said, no proof at all. It is partly that in some cases in which a direct pyramidal tract has been found, there has been weakness of body or increase of reflexes on the non-hemiplegic side. But this proves nothing either, for the affection of the non-hemiplegic side of the body often occurs without any abnormality being perceptible on that side of the cord.

The proof of the variation is then of the most tenuous character, and bearing in mind the objections to it on general grounds, we may safely regard it as purely hypothetical.

And we may go further, and say that in the present state of our knowledge there is little justification for believing that the pyramid fibres end on both sides of the cord. For in certain cases of hemiplegia from cortical lesion, no clinical symptoms can be observed on the non-hemiplegic side, so that we must conclude that the pyramid fibres end on one side only of the cord. When clinical symptoms are observed on the non-hemiplegic side, they are in all probability due to an alteration of the gray substance, brought about by the degeneration of the pyramidal fibres.

*The Posterior Columns.*—Our knowledge about the direct continuation of nerve-fibres of the posterior roots of the spinal nerves into the medulla, we owe chiefly to the observation of Bouchard (1866), of Lange (1872), of Schultze (1883), on man, and to the experiments of Singer (1881) on dogs. Schiefferdecker (1876) was the first to give a detailed description of ascending degeneration after section of the cord in dogs. From these and other observations we gather the following account of the course of the nerve-fibres of the posterior root. Each posterior root on entering the cord forms the most lateral portion of the posterior column, that next the gray substance; a considerable number of its fibres at once enter the gray substance, the rest continue

upwards, becoming, as they go, more mesially situated, in consequence of the entrance of other nerve-roots, and giving off, in their course, fibres of the gray substance of the cord. But not all the nerve-fibres end in the cord; some run on to end in the clava; the course of these fibres has been worked out for the sacral and lumbar nerves only. From what has been said, it is evident that the several posterior nerve-roots form ascending laminae of fibres, so that in the upper lumbar region the nerve-root which has last entered forms a lamina in contact with the posterior cornu, and each nerve-root below this forms a lamina immediately mesial to that of the one above it, the meso-dorsal angle of the posterior column being occupied by fibres from the last sacral nerve. As these laminae ascend in the cord, they give off at intervals, and chiefly in the dorsal region, a considerable number of their fibres to the gray substance of the cord. In the cervical region of the cord all the fibres which have reached it from the lumbar and sacral nerve-roots are found in Goll's column occupying the same relative position as they occupied lower down. These fibres, without much diminution in number, run on to the medulla, and end in the cells of the clava; they form thus an ascending medullary tract.

Hence then, broadly speaking, the nearer the mesial part of the posterior column is to the medulla, the more completely does it become an ascending medullary tract, and the less does it consist of cord fibres simply ascending from the posterior roots. It seems to me probable that it is on this account that in the developing cord—as described by Flechsig—there is in the dorsal cord no area of fibres in which the medulla is developed late, to correspond to Goll's column in the cervical cord. At the same time, one would expect that further observation on the time of development of the medulla of the nerve-fibres would enable the ascending medullary to be distinguished from the ascending spinal fibres in all regions of the cord. Schiefferdecker and Singer with some reservations, and Schultze unreservedly, consider Goll's column to be made up of fibres which come from the sacral, the lumbar, and perhaps from the lower dorsal region. The corresponding fibres of the upper part of the cord they apparently consider run in Burdach's column. Since there are ascending medullary fibres in the lower nerve-roots, we may take it as certain that there are similar fibres and of similar functions in the upper nerve-roots also. On the above view, then, Goll's column contains medullary fibres from the lower part of the body, and Burdach's column contains medullary and spinal fibres from the upper part of the body. The medullary fibres of Goll's column end in the nucleus of the funiculus gracilis; the fibres of similar function in Burdach's column must then end in the cells of similar function to those of the nucleus of the funiculus gracilis; thus they either leave the funiculus cuneatus for the funiculus gracilis, and end in its nucleus, or they end in the nucleus of the funiculus cuneatus; in which case the latter—in part, at any rate—has the

same function as the nucleus of the funiculus gracilis, the one being the centre for the medullary fibres of the upper part of the body, the other for the medullary fibres of the lower part of the body.

Schultze's opinion is based upon an observation in man in which the middle of the cervical enlargement has been completely destroyed; the degeneration of the posterior columns, complete just above the lesion, was in no part of the cord up to the medulla confined in Goll's column, but occupied also a considerable part of Burdach's column. A similar account is given by Löwenthal of the effect of section of the posterior columns in the cervical region in the dog. These observations are not very conclusive as to the point in question; and it may be noted that no one has obtained complete degeneration of Goll's column in the upper cervical region by section of the cord in the lower dorsal region, and further, that Goll's column certainly increases in its way up the cord. We must be content to wait until observations have been made upon the effects of section of the posterior roots of the spinal nerves in the cervical region.

Since the fibres of Goll's column consist of small nerve-fibres, and since there is ground for believing that medullated nerve-fibres do not alter in size in their course in the cord, we may conclude that the medullary fibres of the posterior column consist of a greater or less part of the small fibres of the spinal nerves, and that the spinal fibres of the posterior columns (except perhaps in the upper cervical region) include all the large fibres of the spinal nerves.

The question of the presence of commissural fibres in the posterior columns we may omit, since the recent work on the spinal cord does not deal with it.

*The Cerebellar Tract.*—Singer found, on section of the cord in the dog between the dorsal and lumbar regions, that in the mid-dorsal region, besides the degeneration of the tract ordinarily recognized as the cerebellar tract, there were degenerated fibres scattered over the whole of the antero-lateral column, these fibres being fewest at the periphery of the anterior column. In the cervical region, the degenerated fibres were much less in number; they were present at the periphery of the lateral column only, but stretched from the anterior to the posterior nerve-roots, although they were much more diffuse ventrally of the ligamentum denticulatum than dorsally of it. These fibres in the cervical region were considered by Singer as belonging to the cerebellar tract.

According to Flechsig, the cerebellar tract in man stretches as a compact bundle of fibres about as far as the ligamentum denticulatum; at its ventral end it becomes diffuse, and scattered cerebellar fibres are found for a short distance in the antero-lateral column. Making the fullest allowance for the scattered fibres mentioned by Flechsig, there still remains a considerable difference between his description for man and that of Singer for the dog. The more recent observations on ascending degenera-

tion, both in man and in the dog, have tended to confirm, in the main, Singer's account. That in man the ascending degeneration stretches more ventrally than is described by Flechsig, has been pointed out by various observers, and especially by Schultze. Löwenthal has observed the ascending degeneration in the dog after section of the cord in the cervical region; according to him, the cerebellar tract is fairly compact up to the denticular ligament; beyond this it becomes much more diffuse, but stretches along the periphery of the cord as far as the point of exit of the anterior nerve-roots. Near the denticular ligament, it extends some little distance into the lateral column.

The fact, however, that these ventrally placed fibres degenerate in an ascending direction is alone sufficient to show that they belong to the cerebellar tract. To show this the fibres must be traced up to the cerebellum. According to Löwenthal, the ventral fibres do not run in the restiform body, as do the dorsal fibres, but run in the lateral region of the pons—probably as the “*aberrirende Seitenstrangbündel*” of v. Monakow,—twist round the superior peduncle of the cerebellum, and thence run to the cerebellum.

Löwenthal, like all previous observers, finds that the degeneration of the cerebellar tract diminishes in its way up the cord; that is, that a considerable part of the “*cerebellar*” tract consists of connecting fibres of the cord. Flechsig, on account of the steady increase in size of the cerebellar tract upwards, considered that it received fibres from the cord, but gave none to it; but the fact does not warrant the conclusion, for the tract must increase in size as long as it receives more fibres than it gives off. It may be mentioned that the most of the scattered degenerated fibres—probably connecting fibres—described by Singer in the dorsal region, were not observed by Löwenthal in the cervical.

*Fibres proper to the Cord which undergo Descending Degeneration.*—These fibres are called by Singer, who restricts the meaning of the term, as used by Bouchard, “*commissural*” fibres. The term is not, I think, a very happy one. In anatomy the word “*commissure*” has come to mean the connecting portion between bilaterally symmetrical parts; in this sense fibres which connect the cervical with the lumbar region of the cord do not form a commissure any more than fibres which connect the spinal cord with the cerebellum or with the cerebrum. If “*commissure*” is used in its wider meaning, then all these fibres are equally commissural, the fibres of the pyramidal tract and of the cerebellar tract, as much as those connecting the different segments of the spinal cord. But it would, I think, be better to restrict the term “*commissural*.” The fibres which degenerate can conveniently be designated according to their connection and their mode of degeneration, whether above or below the point of injury. Thus we have ascending and descending spinal, medullo-spinal, ascending cerebello-spinal, descending cerebro-spinal fibres in the cord. But there are many nerve-fibres in the antero-lateral columns

which do not degenerate either above or below the place of section. These might conveniently be called commissural fibres; and the designation would have some justification. The fibres which degenerate have a trophic centre at one end only, and it is very likely that this is due to the nerve-fibres in life conveying impulses in one direction only, for there seems to me no reason to suppose that the fibres of the posterior roots or the fibres of the pyramidal tracts ever receive impulses from the spinal cord. On the other hand, the fibres which do not degenerate on section must have a trophic centre at both ends, and in view of what has just been said, it is probable that this is due to their receiving impulses at both ends. If this is so, these fibres are more properly commissural than those which degenerate.

The proper cord-fibres which degenerate in an ascending direction have already been mentioned in connection with the cerebellar tract, since the two sets have not as yet been with certainty distinguished.

Of the proper cord-fibres which degenerate in a descending direction, those which lie in, and in the neighborhood of, the lateral pyramidal tract have also been mentioned. The remainder are scattered throughout the ventral part of the antero-lateral column. In the dog the longer fibres form a moderately compact zone at the periphery of the anterior column. The observations of Schiefferdecker and of Singer, from whom chiefly the above statements are drawn, were made after section of the cord in the lower dorsal region. In Löwenthal's experiments the spinal cord was cut through more or less completely in the cervical region; he finds throughout the lower cervical, the dorsal, and the upper lumbar region, as far as the lumbar swelling, a degenerated tract of fibres round the whole periphery of the anterior column like that previously described by Schiefferdecker and by Singer, after section of the dorsal cord. But, unlike previous observers, he finds that this tract, at the junction of the anterior with the lateral column, continues dorsally for some little distance into the lateral column, although leaving the periphery and becoming more diffuse. Since these fibres diminish in number in passing down the cord, they must run to different cord segments. Since, further, the zone in the lumbar region is less compact than that previously described after section of the cord in the dorsal region, we may conclude that it receives from, as well as gives off, fibres to all parts of the cord. Some of them probably arise in the medulla, *i. e.*, are descending medullo-spinal fibres.

For the fibres of short course which undergo descending degeneration, Löwenthal finds much the same distribution in the cervical region as that found by Schiefferdecker and by Singer in the lumbar region—viz., in the non-peripheral parts of the anterior column, and in "anterior mixed zone" of the lateral column.

*Tertiary Degeneration.*—Sherrington and myself found that after injury to the brain in the motor area, there was, besides the secondary degeneration in the pyramidal tracts, a degeneration of

a different nature, more resembling chronic myelitis. The altered fibres were chiefly in the anterior columns, varying somewhat in position in different parts of the cord. Since this was apparently the result of an alteration in the gray substance of the cord in consequence of the secondary degeneration of the pyramidal tracts, we called it a tertiary degeneration, at the same time pointing out the possibility of its having been produced by the hardening agent. Sherrington has since found two other cases in which, after a cortical lesion, a similar change had taken place in the spinal cord. As he points out, the entire absence of altered fibres from the cerebellar tract is strong reason for regarding the "myelitic" appearance of certain fibres in the rest of the cord as being due to degeneration, and not to the hardening agent.

*The Parts of the Cortex of the Brain which are Connected with the Lateral Pyramidal Tracts.*—It is well known that a lesion which affects a considerable part of the motor area of the cortex of the brain causes a degeneration in the lateral pyramidal tract of the cord, and that a considerable lesion in other parts of the cortex—such as that of the occipital and temporal lobes—causes no degeneration in the cord.

We know very little more than this, but there has been a great tendency to consider this knowledge as much more precise than it really is. This is so because it is commonly assumed : 1st. That the motor area of the cortex, as it has been determined by electrical stimulation, coincides with the "cord area," *i. e.*, with the area of the cortex, destruction of which causes degeneration in the cord. 2d. That each "centre" in the motor area is connected with its corresponding local centre in the spinal cord, and with that only ; for instance, that the cortical area, stimulation of which causes movements of the fore-arm, is connected with the "arm centre" in the cervical region of the cord, and with no other part of the cord.

Ferrier and Yeo, in a very interesting paper on the physiological effects of extirpation of the cortex in the ape, touch somewhat incidentally on the question of secondary degeneration. In the cases in which the cord was examined, secondary degeneration was only found when the cortical lesion had taken place in the motor area (the convolutions bounding the fissure of Rolando). These observations are distinctly in favor of the view, that the "cord area" coincides with the motor area for the limbs and trunk, but the details given are not sufficiently full to prove that this is the case.

The more recent experiments in the dog are difficult to harmonize with the scheme given above.

Sherrington and myself, from a comparison of the area of greatest degeneration on the two sides of the cord consequent on destruction of the cortex of the brain to unequal extents and at different times, were inclined to believe that the anterior part of the motor area was connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the

anterior part of the lateral pyramidal tract; and further, that the destruction of the cortex behind the motor area as figured by Hitzig and by Ferrier would give rise to degeneration in the cord. The nature of our observations did not, however, allow of conclusions being drawn with certainty. It will be noticed that our tentative conclusions involved: (1) That fibres from each "centre" of the motor area run in a fairly compact bundle. (2) That the cord received nerve-fibres from parts of the cortex which are considered to be motor centres for certain of the muscles of the head. (3) That the "cord area" stretches farther posteriorly than the described motor area.

The posterior part of the cord area—as given above—lies in part, at any rate, in the region which, in Ferrier's observations, produced, on stimulation, movements of the eye. Ferrier considered it, however, to be a part of the "sensory" area of the cortex. This region is called the "Fühlspähre" for the eye by Munk, who puts it in the same class with the cortex of the sigmoid gyrus, which he calls the "Fühlspähre" for the limbs and neck.

Löwenthal has observed five cases in which the coronal and anterior ecto-sylvian convolutions—the anterior parts of the 3d and 2d convolutions—were largely removed; in two of these cases a part also of the median ecto-sylvian and of the anterior sylvian convolution were also removed. In each case, however, a small part of the postero-lateral edge of the sigmoid gyrus was affected; in one case this appears to have been a mere fraying of the edge of the sigmoid gyrus. In every instance there was a degeneration of the lateral pyramidal tract of the spinal cord, a degeneration which could be followed a variable distance in the dorsal region.

If the degeneration was due to the destruction of the coronal, ecto-sylvian, or sylvian convolution, it follows that the destruction of the motor centres for certain muscles of the head, causes degeneration of part of the lateral pyramidal tract of the cord. It is unlikely that the slight and superficial injury to the sigmoid gyrus should have caused so considerable a degeneration as occurred in these cases; but even by granting this, the difficulty is not removed, for the part of the sigmoid gyrus injured is the motor area for the fore-limb, and the destruction of this ought not, on the generally accepted view, to cause degeneration in the dorsal region of the cord.

It may be, however, that in each of these cases the lesion was carried too deep, and affected part of the corona radiata containing fibres coming from the cortical "centre" for the hind-limb. Löwenthal mentions this possibility, but does not say that there was any evidence for it in the sections of the brain.

Sherrington gives an observation which is more difficult to explain on the ordinary view of the connection of the motor centres with the cord. He finds that a lesion which does not reach farther forward than the posterior edge of the ecto-sylvian

fissure, produces degeneration throughout the cord, *i.e.*, a lesion which lies posteriorly to the motor area as given by Hitzig, Fritsch, and by Ferrier,—though including part of the “Fühlsphäre” of the eye of Munk—causes degeneration in the lateral pyramidal tract. In his case it is very difficult to see how fibres from the sigmoid gyrus could have been affected.

Further, Löwenthal finds that lesion of the anterior limb of the sigmoid gyrus—the posterior lateral border being left intact—leads to degeneration as far as the upper dorsal region. No one, I believe, has considered the anterior limb of the sigmoid gyrus to contain a motor area for any of the muscles of the trunk or of the hind leg.

From the destruction of certain parts of the cortex, Löwenthal has observed no degeneration in the cord. In a case in which the lesion was confined to the postero-dorsal part of the sub-orbital lobe degeneration was absent. Sherrington also found no degeneration from injury of the sub-orbital lobe. According to Munk, the sub-orbital lobe is the “Fühlsphäre” of the trunk.

The tentative conclusion mentioned above, that the fibres from different parts of the motor area run in different parts of the lateral pyramidal tract, has received no confirmation. Löwenthal examined the cord in seventeen cases of cortical lesion affecting the sigmoid gyrus in different degrees and in different regions. He mentions no difference in the cord degeneration, except a difference in intensity. Sherrington finds the position of the cord degeneration to be the same whatever the cortical lesion which has given rise to it. If this is so, it shows a very remarkable intermingling of the fibres of the pyramidal tract in their course from the cortex.

A limited intermingling of fibres is suggested by the observations of Hitzig, who found that movements of the muscles of the trunk could be obtained by stimulation of the motor area for the fore-limbs; and a greater intermingling is suggested by the recent observations of Paneth, made under Exner's direction. Paneth finds that there are small motor areas for muscles of the fore- and of the hind-limbs interspersed over a considerable area of the posterior limb of the sigmoid gyrus.

But both as regards this point, and as regards the connection of the part of the motor area with the lateral pyramidal tract, we must wait for further investigations. The experiments which have been mentioned above suggest conclusions which are not in accordance with those which we should on general grounds be inclined to accept; for this reason, then, we are justified in reserving a definite opinion until fuller and more satisfactory evidence is forthcoming.

---

**Embolism of the Medulla.** By GEO. B. SWASEY, M.D.  
*N. Y. Med. Rec.*, August, 1886.

Dr. Swasey reports an interesting case of what he considers to

be "Embolism of the Medulla." The main points in the history are as follows : J. D. Warter, æt. thirty-two, single, heavy drinker had chancre three years ago. Evening of April 9th, drank freely ; next morning found that he had lost power of speech, unable to swallow, and had pain in region of sternum ; was dizzy and staggered ; ringing in his right ear. Pulse that same day, 108 ; temperature, 99.5° F. Some paresis of lower extremities, most marked upon right side. Could walk quite well with eyes closed, and could stand with heels together without reeling. (This in the afternoon.) Stood on one foot with eyes closed, but with much difficulty upon the right owing to its weakened condition. There was some paresis of thigh muscles, more marked on right side than on left. He was able to open his mouth over half the distance, but was unable to protrude his tongue ; mucous membrane of palatine arch was anæsthetic. Facial nerve upon right side was paretic, most marked on its lower fibres of distribution ; left facial less paretic than right. Respiration, 22, "slightly Cheyne-Stokes." Frequent gaping. Six days later : Temperature, 98½° ; pulse, 94 ; respiration, 16. Paresis of both forearms, greater on right side. Plantar reflexes lessened ; left cremaster lessened, right absent ; patellar reflexes exaggerated ; left ulnar and radial reflexes present, right absent ; sensibility normal, bladder normal, bowels constipated. In the course of a month or two all symptoms disappear, with the exception of occasional difficulty of speech and some slight trouble in holding his water. Patellar reflexes exaggerated, left more so than right. Both radial reflexes normal<sup>1</sup> ; others not tested. The author believes that an embolic obstruction occurred in one of the vertebrals, but he is not willing to decide whether upon the right or the left side. The writer makes out a fairly strong case in support of his diagnosis. We do not feel quite so certain that this is a case of medullar disease. The amount of paralysis that existed was slight, and neither respiration nor the pulse was at all seriously interfered with. The chief symptoms were those of aphasia and hypoglossal disease. We ask the writer's attention to a paper by Edinger reviewed in this JOURNAL—page 256, of this volume. B. S.

### Symmetrical Vaso-Motor Paralysis of Both Hands.

*Gazette des Hôpitaux*, p. 501, 1886.

The patient, male, æt. fifty-four, entered the hospital on the 1st of May. No hereditary history. Health generally good ; never had any acute disease ; no tendency to nervous affections ; well nourished. Without any assignable cause, the patient, four years ago, at the beginning of the winter, noticed that his hands were swollen ; this swelling, which was not painful, lasted a fortnight. Five months ago the same swelling, again without pain, returned. Upon examination both hands were found red, hot, and swollen. The skin is thick and hard and immovable. The dorsal surface

<sup>1</sup> Present or absent (?—Reporter).

is somewhat œdematous, and the veins are enlarged. The lower part of the arms is somewhat hot, otherwise the arms are normal. Appetite good, but a certain amount of polydipsia, with corresponding polyuria. No sugar in the urine, but slight traces of albumen. Heart-sounds normal. The axillary temperature is  $37.4^{\circ}$ , that of the forearm  $34.6^{\circ}$ , of the dorsum of the hand  $35.5^{\circ}$ , of the palm  $35.6^{\circ}$ , and between the index and middle finger  $35.8^{\circ}$ . The œdema began to disappear after a few days, and after three weeks the hands were again in their normal condition. The diagnosis in this case wavered between an œdema due to a disorder of the kidneys or heart, erythema, acrodynia, rheumatism, and the one which was made of vaso-motor paralysis.

---

**Poisoning by Carbonic Oxide—Paralysis of the Extensor Muscles of the Left Forearm—Rapid Cure.** SCHACHMANN. *La France Médicale*, July 1, 1886.

Various theses published within the last ten years have again called attention to the influence of carbonic oxide gas upon peripheral nerves. The subject was not new, but forgotten, for as early as 1843 Bourdon minutely described the accidents due to the action of this gas. The number of well-observed cases is not large. S. here describes the case of one of three persons who attempted suicide together by asphyxiation with this gas. Two of them succeeded in the attempt, and one survived.

B. C., female, æt. twenty. Family affairs led her to attempt suicide. The attempt was made on the 8th of May. All the apertures of the room were carefully closed, and the open stove with charcoal was lighted. She was discovered in an unconscious state and taken to the hospital.

May 10th.—Patient seems to have entirely recovered from the effects of the gas, but upon examination a complete extensor paralysis of the left forearm is found. Also about the middle of the arm a now fluctuating swelling is found. No electrical examination of the muscles.

May 11th.—Condition of paralysis and swelling the same.

May 12th and 13th.—No change.

May 14th.—Slight movements of extensor with fingers. Diminution in size of swelling.

May 15th and 16th.—Improvement.

May 17th.—Almost complete restoration of functions.

May 20th.—Complete cure.

(The author's theory that the paralysis was due to the action of the carbonic oxide gas is certainly not proven by the history given. The general impression left is that of a traumatic radial paralysis.)

G. W. JACOBY.

---